ON EXCESSIVE SENSORY CORTICAL DISCHARGES AND THEIR EFFECTS:

A STUDY OF SENSORY AURÆ.

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ON EXCESSIVE SENSORY CORTICAL DISCHARGES.

THE expression "cortical discharge" will in the present communication be understood to mean a sudden liberation of the functional activity of any portion of the cortex cerebri. Assuming a certain degree of this to be a normal phenomenon, its intensity may be increased or diminished under a variety of morbid conditions. The first of these modifications—increase—forms the subject matter of this paper.

Excessive cortical discharge is consequent upon a state of hyper-excitability of the cellular elements of the brain caused by their irritation, which irritation may either be induced by mechanical, chemical, electrical, and other artificial agents, or by diseases of various kinds. This internal central change generates a corresponding series of external manifestations, which take the form of explosions or sudden exaggerated paroxysms of that special function which is represented by the affected central grey matter. For example, if the motor area of the cortex be involved, a convulsion results; if the sensory region, a violent subjective storm of one or more senses. A "discharging lesion" therefore is the state of instability of cortical cells, which in its turn may be due to a variety of pathological causes. The "discharge" itself is the sudden, excessive, concentrated

liberation of their function, which as a consequence follows. The "symptoms of the discharge" are the paroxysmal exhibitions of excess of action of different kinds, in different regions by those elements throughout the body whose functions are represented in the centres which are involved by the disease.

All nervous action may be regarded as dependent on nerve cell activity, the product of which constitutes that which, for want of a better term, is called nervous force or energy. This is generated during the normal processes of nutrition, to be expended in initiating and maintaining the complex requirements of animal life. Nerve cells, besides generating force, possess the inherent property of resisting its waste, so as to permit, up to a certain point, of its accumulation in their interior. Hence they may not only be regarded as manufactories, but as magazines, for this commodity. From thence as occasion demands, nerve force is set free in different forms and through various channels for manifold purposes, having overcome the natural resistance which so far had acted as a restraining power in preventing its wanton dissipation. Under normal conditions there is a constant relation between the generation and storing up of this energy, and the resistance in the cell elements to be overcome during its liberation, the mean between the two constituting a state of healthy nervous stability or equilibrium, the supply of the former and the influence of the latter being in proportion to one another. Like a charged galvanic cell, the nervous ganglion is in a condition of latent energy, requiring only a liberating agent to set free its power, the adjustment between the active force produced and the resistance restraining it being so equable, that the most delicate controlled and harmonious manifestations are possible. Normal nervous action may be summed up as the product of normal nerve force liberated

through normal resistance. The whole question, therefore, of cortical discharges in health or disease is centred on the integrity of this law, and their character depends entirely on the relation which exists between force and resistance. If from any cause the first is in excess or the latter is diminished, the normal balance is overthrown, and in consequence the functional activity which is set free is increased in quantity, and possibly altered in quality. It is the morbid explosion of augmented energy that results from either of these conditions, which is here indicated under the designation of excessive discharge, which may be regarded as a simple exaggeration of a natural process in which either the force produced is excessive, or the resistance interposed is diminished. The reverse of this—namely, diminished force or increased resistance—would obviously have opposite effects; but with such phenomena we are not at present concerned.

The most common conditions we know of under which paroxysms of increased nerve energy take place is when there is a hyper-physiological functional activity of the cellular elements as a result of their irritation. The excess of action which as a consequence ensues overcomes the natural resistance of the tissues, the healthy balance between the two is upset, and leakage or overflow of function is the result, in the shape of excessive, unrestrained explosions of energy, concentrated alike in amount and in time of production. A continuous excitation, for example, of the cortex cerebri augments the special functional activity of its cells. If the stimulation is not too severe, this effect accumulates up to a certain point, and, when the natural elastic limit of resistance in the restraining elements is reached and overcome, an explosion or sudden discharge of functional activity takes place, constituting the attack or seizure. If the irritation is excessive, the results are the

same, only they are more rapidly developed and more widely spread. Little definite is known of increased nervous discharge due to diminished resistance alone, but it is highly probable that many of the explosions of function and paroxysmal seizures met with in disease depend upon this condition.

Given an excessive nervous discharge, both theoretical and practical considerations seem to suggest that its character depends on certain physical conditions which may be briefly summarised as follows. The *intensity* of the effects of an explosion of nerve force depends on the amount of resistance, rather than on the quantity of the irritation. The greater the resistance offered to the liberation of energy, the more violent will be the resulting effects. The reason of this is that, in order to overcome the increased obstruction, force must accumulate until it has reached the necessary point of tension; hence its severity when exploded. Thus mere quantity of paroxysmal display is independent of quality of excitation, as, if this last be small, it becomes only a question of time to amass sufficient force to overcome the increased difficulty. To put it in another way, resistance being constant, the effects of irritation, whether great or small, will always be the same in degree. For the same reason, the less the resistance, the milder will be the effects of discharge. The *frequency* of the explosions, on the other hand, chiefly depends upon the amount of the irritation, assuming the resistance to be constant. The more intense the excitability, the more rapidly in point of time do the paroxysms occur, and, *vice versâ*, it is apparent that a strong irritation will overcome a given resistance more quickly than a slight one, which will take a longer time by accumulating to arrive at the necessary tension to effect the same end. On the other hand, for a like reason, irritation being the same, a slight resistance will be overcome more rapidly than a great one,

and the explosions consequently will be more frequent. The *distribution* of the effects of cortical discharge depends partly on the intensity of the irritation, and partly on the state of the resistance. In the first place, normal nervous influences will always most readily follow the most accustomed paths, and these "in the order of their speciality," and therefore those of least resistance. When a discharge is excessive, greater resistance is overcome and a larger number of channels are opened up, and thus the effect of the increased explosion of energy is wider spread. On the other hand, when a discharge is slight, its influence is limited to the paths of least resistance; hence the effects are more local. In the second place, with a given irritation, the results would be extensive or restricted according to the greater or less resistance of the nervous elements. Of this, however, we have little practical knowledge, as we cannot formulate the laws which govern the resistance of the nervous elements in disease. There can, however, be little doubt that the changes in this respect must vary greatly in different morbid conditions, and must be a potent factor in the severity and distribution of cortical discharges and their effects. In an ordinary epileptic seizure, if the amount of irritation could be measured, knowing the distribution of its effects, we could gauge the resistance of the nervous tissues; or *vice versâ*, if we knew the resistance, we could calculate the amount of irritation. In disease, however, both of these are obviously beyond our powers of estimation, although the great variety of degrees of a convulsion are doubtless due to some alteration in the relative proportion of one or other of these factors.

In all cases, whatever the intensity, frequency, or distribution of explosions the result of an excessive discharging lesion may be, the constant characters are *suddenness* and

periodicity. That an excessive cortical discharge and its effects should be sudden follows from what has already been said concerning the properties of the ganglion cells with regard to force and resistance. When a continuous force overcomes a continuous resistance, the resulting liberation is intermittent and paroxysmal in character, owing to well-known physical laws. The same takes place when vital elements come into play, and all exhibitions of nervous energy are of this periodic type. In the normal state every ordinary movement or muscular contraction may be analysed into a series of smaller movements or contractions, the rapid succession and aggregation of which constitute what is apparently steady coördinate action. Excessive functional activity, or an exaggeration of healthy processes, and morbid increase of energy, present the same characters, being intermittent in composition and occurring in paroxysmal sequences. For the production of the last the cells become overcharged, and, as a consequence, explode; a period of rest ensues, shorter or longer, according to the amount of excitation, during which fresh accumulation goes on, again to repeat the cycle of events. The period of actual discharge, liberation of force, and resistance overcome, alternates with the period of rest during which production, accumulation, and storing up goes on. This constitutes an essentially periodic process. The external results, or the symptoms, the exact nature of the paroxysms which are developed, depend on the function of the particular cortical cells involved, and may consist of a great variety of motor, sensory, or intellectual phenomena. They may be produced artificially by experiment, and are caused by disease, as typically seen in epilepsy and epileptiform attacks.

Should these abnormal discharges of nervous force be excessive either in degree or duration, and especially the former, exhaustion of the cell elements results, and *tem-*

porary paralysis of their function as a consequence ensues. That every form of nervous functional activity is succeeded by a corresponding reaction of depression may be admitted as a general law. In health, if not excessive, this is not apparent, as the ordinary processes of nutrition conceal its effects. Here there is always a relation between the amount of positive liberated action and the subsequent development of the negative condition of fatigue, the one being in an inverse ratio to the other. Up to a certain point this is a normal state constituting healthy fatigue after exertion, which is restored by appropriate rest. The limit to which the normal nervous system can perform work and withstand its effects depends upon its inherent and individual vitality. If this boundary be passed, if the expenditure of force exceeds the convenient demand, exactly in proportion will fatigue and functional incapacity be produced, which will require a corresponding time for recuperation to take place. The functions of the cortex cerebri in every way obey these general principles; and common experience shows that when motor, sensory, or intellectual efforts are made, the cells which represent these faculties undergo proportionate fatigue, and require time and rest for their renovation. The power of production, the resistance to depression, and the capacity for recuperation vary in different individuals according to the functional stability of their respective nervous systems. Any alteration in this natural process by disease or otherwise further emphasises these truths. Direct experimental irritation of any portion of the cortex first produces an excessive display of its special function, and this is followed in the same ratio by a corresponding depression. For example, a continuous electrical stimulation of the motor cortical area representing the arm will cause a convulsion of that limb. Gradually, as the centre is exhausted, it becomes insensitive to stimulation,

and a condition of paralysis follows in the same muscular distribution that was previously convulsed. The completeness of the paralysis is in direct proportion to the severity and duration of the convulsion. After rest the parts gradually regain their natural condition. The cells are first in a state of hyper-physiological excitability, and afterwards in exactly the opposite condition, each stage being accompanied by appropriate symptoms. Irritation from disease causes precisely similar results, which may be seen under a variety of circumstances, but notably in epileptiform seizures. In disease, however, a new element is introduced which may disturb the exact relative proportions between the primary excess and secondary depression of function. Here the nervous system itself is morbid, the tissues are altered, and the anatomical and physiological properties of structure are changed. Hence the natural balance between the effects of excitation and its sequences may be deranged. The vigour of the nervous elements being impaired and their recuperative powers enfeebled, they fail to obey those laws which naturally guide their actions. On this account, the proportion between the convulsive effects of irritating cortical lesions and the consequent paralysis may not bear the classical relation they would do in the simple exaggeration of the normal state. Of the exact influence of diseased cerebral structure in this relation we know little or nothing; but it may go far to account for the apparent contradiction that, in epilepsy more especially, the violence of the convulsion is not of necessity proportionate to the succeeding paralysis. Although this discrepancy may be admitted in a diseased state of the cortex, the relation between the two even here is very close, and no one has insisted on this more than Dr. Hughlings Jackson. He maintains[1] that post-epileptic and epileptiform para-

[1] Brain, Jan. 1887.

lysis is dependent on, and is proportionate to, the amount of severity of the cortical discharge. He considers that the greater and more rapid this is, the more widespread and the more transient the paralysis. On the other hand, the slower and less intense the discharge, the more local and persistent will be the paralysis. Whence practically a diffused and temporary paralysis, which is often overlooked, is simply a local and more persistent one "thinly spread out." Dr. Jackson believes, in short, that, taking severity, rapidity, and distribution into account, there is a true proportional ratio between the discharge and the paralysis which follows it. Even if objections may be raised against this hypothesis—namely, that after severe convulsions there is apparently little paralysis, and that after slight spasm this may be considerable—there always remains the fact that from discharging lesions there result convulsions and subsequent paralysis, the two bearing a certain proportion to one another. If the relation in all cases is not exact, it may be explained by the morbid state of the cellular elements, which in some way unknown modify the general law which holds good in health.

Modern science, as the result of experiment, clinical observation, and pathology, may be admitted to have established the fact that there are localised cortical centres which in *motor* function correspond to different regions of the body. It has been abundantly proved that irritation of certain areas, whether produced artificially or by disease, induces well-defined movements in definite muscular distributions; and thus, when the same areas are destroyed, voluntary paralysis results in exactly corresponding regions. Not only has the association with certain areas of the cortex with the separate limbs and large physiological groups of muscles been demonstrated, but in certain instances a relation has been determined between minute portions of the grey

matter and very limited portions of the body, and even single muscles. Doubtless, as our knowledge advances, this connexion will be further differentiated. A study of epileptic seizures shows that a widespread irritation is followed by general convulsions, which, if excessive, are succeeded by a condition of temporary general muscular debility; if the excitation be local, there is a corresponding limited muscular spasm succeeded by the opposite condition of paresis in the same region. A sufficient number of cases have been recorded to prove that this irritation and consequent discharge, and its effects, may be confined to very limited areas of the cortex, and, as a consequence, that the symptoms are restricted to individual limbs and small groups of muscles.

Although this association of definite limited areas in the cerebral cortex with corresponding local regions of the body is recognised and generally admitted as regards the motor functions, the same cannot be said of the different varieties of *sensation*. A vast amount of labour has been spent in attempting to determine the localisation of the sensory centres; but although much instructive experience has been gained, there is as yet little unanimity of opinion as to the conclusions established. There are, for example, conflicting assertions as to the locality of the sense of touch, and some observers even deny the possibility of its limitation to any special region. Certain physiologists believe they have determined the areas in which the special sense of sight, hearing, smell, and taste, centre, but their conclusions are negatived by other observers equally deserving of credence. The reason of this divergence of opinion on the question of the localisation of the sensory functions is not far to seek. The difficulties in the inquiry are enormous. Motility is comparatively a simple phenomenon to determine, and can be experimentally demonstrated in animals. Sensibility is a far more complex

subject, which by the reflex movements artificially produced in the brute creation, can only be very imperfectly inferred. For obvious reasons this cannot be ascertained by experiment in the human subject, who alone could satisfactorily solve most of these problems. We are therefore mainly dependent upon the symptoms of disease in man for information on this subject, and these, in conjunction with pathology, must be our chief guide in investigating this question. It has always struck me that the sensory symptoms which often precede and accompany an epileptic seizure might be made use of in this direction, and it is for this purpose that the present inquiry has been undertaken. The following observations, if they do not serve to map out definite anatomical areas in the cortex representing the special senses, at least suffice to prove that each of them is represented in definite regions of the brain, and that they obey the same general physiological laws as the motor functions. In order to investigate this point, I have made a very careful inquiry into a large number of cases of epileptic and epileptiform attacks, and have accumulated certain facts which seem to throw light on the subject. Just as on the motor side there are explosions of the whole or part of the cortical centres with corresponding general or local convulsions, succeeded by temporary exhaustion of the centres and corresponding muscular paralysis, so I find, on the sensory side, there are analogous phenomena. Here, also, there may be explosions of the cortical representatives of the different forms of sensation, causing exaggerated displays of function—or, as they are termed, crude subjective sensations—followed by temporary abeyance of the same. This would seem to indicate that there are special central regions associated with every sense; that each of these may be separately affected by disease; and that these are capable, when irritated, of discharging an accumulation of

special function. Moreover, they in consequence become subsequently exhausted, as evidenced by the temporary abolition of the sense which had previously been in excess.

With the view of illustrating this position, I propose to pass in review the phenomena observed in a series of cases which have come under my personal observation. For the present, I shall confine my investigations to the five special senses—namely, those of touch, sight, hearing, taste, and smell,—leaving for some future occasion consideration of the equally interesting problems connected with the intellectual faculties, with the many forms of alteration in speech, and the various other sensory disturbances of the different organs. In dealing with sensory phenomena and the five special senses, I have limited myself to the discharge of what Spencer and Jackson call "crude sensations," or the development of simple subjective sensations of pain, colour, noise, taste, and smell; and do not discuss the more complicated phenomena connected with each of these—such as the hallucination or delusion of figures, faces, music, the "dreamy state," and so on, all of which have probably a different signification from that of simple cortical discharge. My experience is derived from a careful analysis of 500 consecutive cases of epilepsy and epileptiform attacks under my own care and investigated by myself. In few of these was there a post-mortem examination, but the symptoms observed speak for themselves. No exact statistical method is attempted, as those who deal with epilepsy will recognise the difficulty and uselessness attendant on such a method of investigation. In most instances the physician has to rely upon the statements of the patient or his friends, having rarely himself the opportunity of actually witnessing the seizure; but in this inquiry every effort has been made, by the strictest cross-examination, to elucidate the facts. I have been fortunate enough in a few but important

exceptions to have actually been present at some of the attacks, and to have personally tested and confirmed the previous statements of the patient. As a matter of convenience I shall take the consideration of the five special senses in the order of the frequency with which they are affected — namely: (1) Touch, (2) Sight, (3) Hearing, (4) Taste, and (5) Smell.

Sense of Touch.

Under the sense of touch it is proposed to consider those cases of an epileptiform nature in which there are paroxysmal attacks of "sensations" of various kinds which are followed by a period of more or less "numbness" or loss of sensibility in the same distribution, and these apparently limited to the integument. These sensations vary greatly in character, consisting often of indefinite feelings, such as crawling, pins-and-needles, itching, and so on, in all degrees up to severe and intolerable pain. These are sometimes limited to one particular spot or region; sometimes they begin at one point and spread in various directions, and sometimes they are more diffuse, and occupy the entire half or even the whole body. These "sensations" are often very difficult to differentiate from those associated with motor phenomena, such as cramps and spasms or the true motor aurae. In the present instance care has been taken as far as possible to eliminate such cases, and to include only such feelings as apparently affected tactile sensibility of the skin, apart from any motor manifestations. In 500 cases of epilepsy or epileptiform seizures there were forty-nine in which the aura appeared to be an uncomplicated modification of cutaneous sensibility. In illustration, the following selected cases may be given, each of them indicating some special peculiarity.

A man aged fifty-one has for many years suffered from ordinary epileptic fits, accompanied with loss of consciousness and general convulsions, always preceded by a sensation in the right arm. Once or twice a week he has slighter attacks of the following nature. Suddenly he is seized with a peculiar sensation, almost amounting to pain, in the fingers of the right hand; this rapidly spreads up the arm to the right side of the face and down the right leg. He is a little confused, pale, and giddy, but does not lose his senses. This condition lasts three or four minutes and then passes off, leaving a sensation of numbness in the affected parts. On testing the skin of the right side during this period, it is found anæsthetic and analgesic, although not completely so. In the course of an hour or two this gradually disappears, and the integument returns to its natural condition. Otherwise the man's condition is normal, as is also his cutaneous system, and no convulsion or motor phenomenon attends this seizure. This is evidently an abortive epileptic attack, and entirely limited to the sensory system. It suggests an explosion of the centre of common sensation on one side, as evidenced by the sudden paroxysm of pain and the subsequent exhaustion of the same, as shown by the temporary loss of sensibility in the same distribution.

A man aged thirty-eight has frequent epileptic seizures, with loss of consciousness and general convulsions. For a minute or two before he loses his senses he feels a peculiar "prickling" all over his body, which almost amounts to pain. After the attack he states that on recovering from temporary drowsiness he is very weak, and the whole of his skin is numb and insensitive to touch. This I had no opportunity of personally testing, but his wife, a very intelligent person, assured me that for an hour or more after the fit he is unable to feel a pinch or rough manipulation of his skin, a fact which she has many times tested.

This case suggests a general explosion of all the intellectual, motor, and sensory centres, as evidenced by the sudden attack of loss of consciousness, widely spread convulsions, and general pain, followed by temporary drowsiness, general muscular weakness, and universal anæsthesia.

A man aged twenty-nine, after a blow on the head received some years ago, has had attacks of the following nature. He is suddenly seized with a violent pain in his left foot, which makes him cry out. This runs up the leg to the trunk, and lasts for a minute or so. He does not lose consciousness, and has no convulsions of any kind. Shortly afterwards his left lower extremity "has lost all feeling," and, on testing it, it is found completely anæsthetic and analgesic. These attacks occur several times a day, and during the intervals the limb never entirely recovers its sensibility. This seems to be a cortical discharge, limited to the leg sensory centres on one side.

A woman aged twenty-six has had several times a week for the last eighteen months attacks of the following nature. A creeping sensation begins in the right forefinger, spreads to the other fingers and thumb, and runs up the arm over the breast and shoulders to the right side of the face, mouth, and tongue. Her power of speech is arrested, but she does not lose her senses, there being only giddiness and some confusion of intellect. This condition takes place slowly, some minutes elapsing before the entire round is completed. There are no convulsions of any kind. Afterwards there is numbness over the arm, right upper half of the body, and half of the face and tongue. (This was repeatedly tested, the anæsthesia being only partial.) This passes off in an hour or two, leaving the skin normal.

A man aged thirty-six has frequent attacks of a sudden pain in the right side of his face and tongue. He loses all power of speech; he is confused and giddy, but does not

lose his senses, and has no spasm or convulsion of any kind. After "a few minutes," he says, the painful sensation goes off, and for some hours afterwards the right side of his face is "numb and dead." I had no opportunity of testing this case, but the description of the man seemed intelligent.

Such cases, with varying details, might be multiplied. I have given a few of the illustrative examples. In the majority of instances the physician has no opportunity of testing the facts, and has to depend upon the statements of the patient or his friends. Of the preceding examples I personally examined the patients during the attack in all except two, and can answer for the accuracy of the description of the symptoms. Of the forty-nine cases in a total of 500 where aurae were apparently of a cutaneous sensory nature, the following is a rough statistical statement of the different regions of the body affected :—

Sensation beginning in one limb and extending over entire side	21
,, all over body	13
,, in face	5
,, limited to one arm	4
,, limited to one leg	3
,, in one side of face and tongue	1
,, in head	1
,, in one hand	1

Besides the foregoing cases, in which sudden paroxysmal attacks of sensation or pain, followed by temporary anæsthesia, appeared to be limited to the skin of the limbs or a larger portion of the body, there are those in which the same phenomena seemed to be limited to the tract of a single nerve. As illustrations of this class of case the following may be cited.

A lady aged fifty was sent to me as suffering from facial neuralgia. For years she had been attacked almost daily, and often several times a day, with sudden seizures of severe pain affecting all the three divisions of the fifth

nerve. The general health was fairly good except a depressed state as the result of prolonged suffering. The seizure was accompanied by pallor, sickness, giddiness, and slight confusion of ideas, but no loss of senses or motor phenomena of any kind. Suddenly the patient would be seized with a violent excruciating pain, which caused her to cry out, affecting the whole of the left side of the face, and especially in the areas of the three divisions of the fifth nerve, which, during the attack, were painful to the touch. This lasted for perhaps from ten to twenty seconds, and caused the greatest agony. This slowly (i.e., in from ten to twenty seconds) disappeared, leaving a feeling of numbness in the same distribution where formerly the pain had existed. Repeatedly tested, there was found to be partial anæsthesia of the parts, which remained for several hours and then gradually subsided, the face returning to almost a normal condition. All remedies directed towards a neuralgic theory having had no effect, thirty-grain doses of bromide of potassium were administered thrice daily. The effect was immediate. At once the attacks were arrested, and instead of one or more attacks per day, they were reduced to one or two a month, and even these were of much less violence than before. The patient continued in this condition for several months, when she was unfortunately lost sight of. I think there can be little doubt that this was a case of true sensory epilepsy, the nature of the symptoms suggesting this theory, which was further supported by the results of treatment. Subsequent inquiry ascertained that this lady had suffered from ordinary epilepsy in her youth. I have seen several other cases of almost exactly similar character. May this not be an explosion of that portion of the cortex which represents the distribution of the fifth nerve?

A man, aged forty-five, eighteen months ago had an abscess at the back of the right thigh near the hip-joint.

A cicatrix three inches long is seen at the back of the thigh, over and in the line of the sciatic nerve. The patient says that the surgeon who treated him and incised the abscess told him that the sciatic nerve was exposed and injured. Some months afterwards he had attacks of violent pain in the tract of the right sciatic nerve and also in the foot, lasting for some seconds, accompanied with convulsion of the limb, but without loss of consciousness. I had no opportunity of personally testing this patient, as I never witnessed an attack, but the man was very intelligent, and stated that immediately after the attacks, two or three of which occurred weekly, the limb was almost entirely paralysed for an hour or so, and he could not walk. At the same time "it lost all feeling, so that he could pinch it without any sensation being excited." Both of these symptoms almost, but not entirely, disappeared in a few hours. On examination I found slight motor paresis of the limb, and also slight loss of sensibility of the skin of the foot and leg below the knee, which it appears is his usual condition. Here also this would appear to be a case of sensory epilepsy limited to the distribution of the sciatic nerve, complicated, however, with a motor epilepsy as well.

I believe that such cases of sensory epilepsy, limited to the distribution of special nerves, are more common than is generally recognised. They are usually confounded with simple neuralgia or spasmodic tic. Doubtless many persons thought to be suffering from neuralgic affections, migraine, angina, sciatica, &c., are in reality the victims of epileptiform seizures, and are to be classed under this category, and not as local nerve lesions. A recognition of this fact and a differentation between the two is obviously of the greatest importance as regards the prospects of prognosis and treatment.

There are many other interesting and important problems connected with discharges of other portions of the sensory cortex which it is not proposed to discuss here, as for the present I confine myself to a consideration of the five special senses. Explosions may occur in the intellectual centre, giving rise to what Dr. Hughlings Jackson calls "the dreamy state." There are also various visceral sensations associated with epileptic attacks. There are the phenomena connected with speech, and doubtless many other sensory disturbances, which, however, are not included in the present inquiry.

These observations on the paroxysmal disturbances of cutaneous sensibility do not, of course, throw any light on the anatomical localisation of a centre for the sense of touch. But I think they serve to indicate that there must exist certain areas in the cortex cerebri associated with the tactile sensibility of the different regions of the body. These may be diseased in their entirety or in part, as it has been seen that quite limited portions of the skin, and therefore presumably of the cortex only, may be involved. This would seem to suggest that the function of sensibility in its distribution to the different regions of the body, like that of motion, is specially localised in corresponding areas of the cortex, and that one or more of them may be affected by disease, leaving the others normal. Further, this inquiry points to the conclusion that when the sensory centres are in a hyper-physiological state as the result of irritation, they obey the same laws as the motor ganglia, by paroxysmally discharging, as it were, their respective functions, and that when in consequence they are exhausted, the opposite effect, or paralysis, ensues.

Sense of Sight.

After that of touch, the sense most frequently affected by the epileptiform condition is that of sight. In 500 cases of epilepsy there were sixteen in which some peculiarity connected with the function of vision was the prominent feature. Doubtless in many others some ocular symptoms formed a part of the attack, but in sixteen only may it be said to have been the leading and essential phenomenon. The seizure consists of a sudden display of colours or light subjectively seen, and described by the patient. The most common is red, but occasionally there is a play of different hues like a spectrum. Sometimes the light sparkles, flashes, and scintillates; more commonly it is described as being of a brilliant and more or less continuous character. It usually lasts from a few seconds to a minute or two, and then quickly or gradually disappears. Sometimes this is referred to one eye; more commonly to both. It has been impossible to determine accurately to what portion of the retina it is limited. This appearance of light, in the few instances in which there has been an opportunity of testing the question, is followed by amblyopia or confusion and deficiency of vision. This may be limited to one eye; more commonly it exists in both. In the latter case, in at least one instance, it was found to consist of homonymous hemianopia. This amblyopia is usually very temporary, lasting sometimes only a few minutes, occasionally longer. These amblyopic conditions are very difficult to determine with accuracy. The physician rarely has an opportunity of being present at the actual attack, and their fleeting character prevents their being measured with precision. The few cases which I have had the good fortune to personally investigate merit briefly some detailed notice.

A man aged thirty-six received a blow on the right

parietal region six years before he came under observation. The details of the first portion of his case have been published,[1] and need not be repeated here. It is sufficient to state that for some years he had been subject to epileptic attacks of a very violent nature. These are always preceded by the appearance of a bright-red light, he thinks confined to the left eye, following which he has a general convulsive seizure. He was trephined over the right parietal bone, after which there was no return of the attacks for ten months. Subsequently these again returned in a form which was limited to the visual aura. Suddenly he would be seized with a feeling of fulness and confusion in the head, pallor, and a bright-red light in the left eye. This would last from thirty seconds to a minute, without loss of consciousness, and then disappeared. Immediately afterwards the sight in the left eye was found greatly impaired. He could see light, but could not count fingers, and this deficiency extended over the entire field of vision. This condition lasted from a quarter of an hour to nearly an hour, when the eye gradually resumed its normal condition. Ophthalmoscopic appearances were normal. These facts were repeatedly tested by myself, as he had many such attacks every day, and moreover they could be produced by pressure over the tender cicatrix. The patient was trephined again, and the painful tissues removed, after which there was no return of the seizures, while the patient remained under observation for several months. This case seems to me to suggest the following considerations:—1. An injury over the neighbourhood of the right angular gyrus was the apparent cause of the attacks. 2. The lesion was of an irritating character and caused a discharge of visual function, as evidenced by the sudden paroxysmal subjective display of red light in the left eye. 3. The effects of irrita-

[1] Brit. Med. Jour., Jan. 1st, 1887.

tion were followed by those of exhaustion, as manifested by amblyopia of the entire field of the same eye. 4. Affection of a certain area on one side of the brain produces its effect on the opposite eye only. 5. Trephining by the removal of the cause of irritation arrested the phenomena.

Along with the above must be taken the following case, which is also of much interest, as showing a totally different effect on vision from cortical disease.

A man aged forty-eight had been for a year paralysed on his left side, which was also in a state of constant tremor. He was subject to occasional epileptic attacks of a severe general nature, but had frequent seizures of a slighter character. These last consisted—in addition to headache, giddiness, and some mental confusion—of the sudden appearance of a bright-red light *in both eyes*, the position of which could not be exactly defined. There was no loss of consciousness. In less than a minute this passed off, leaving a deficiency of sight in both eyes. On careful testing, it was found that homonymous hemianopia, of a not complete type, existed on the right side, so that the two halves of both left fields of vision were nearly blind. This disturbance of sight was only computed by rough testing with the fingers, as, owing to its temporary nature, there was no opportunity of more accurately measuring the condition into a perimeter. There was, however, no doubt of the general fact, which was observed on several occasions. After death extensive softening was found of almost the entire cortical motor region on the right side, which involved the angular gyrus and almost reached as far as the occipital and temporal convolutions. This case is quite different in its results from the preceding one, but is of equal interest. It seems to show that irritation of some other portion of the cortex causes a discharge of light, which is attributed to both eyes, and that the subsequent

exhaustion of the centre had for its result temporary homonymous hemianopia on the same side as the lesion. The following is probably another case of the same kind.

A woman aged twenty-one has for many years been subject to epileptic attacks, with general convulsions. She says that each fit is always preceded by "flashes of different colours like a rainbow" in both eyes. When she recovers consciousness, she asserts that, although sensible, she is unable to see objects for nearly an hour, after which her sight gradually returns. This patient I had no opportunity of personally examining during an attack.

In nearly all the cases where the aura consisted of subjective sensations of light, the patients on interrogation asserted that after the attack there was more or less disturbance of vision of a temporary character. It was found impossible from the description to ascertain the exact distribution either of the irritation or the paralytic symptoms. Indeed, in most cases the individual is incapable, under the circumstances, of localising them by themselves, and it is extremely rare for the observer to be present when an attack takes place. No reference is here made to other forms of visual aura which are sometimes met with, such as indefinable appearances before the eyes, temporary loss of sight, occurrence of figures and objects, word-blindness, and so on. The present inquiry is limited to those cases in which there seemed to be evidence, first, of a discharge of crude visual function, as evidenced by a sudden paroxysmal display of colour, followed by a temporary abolition or a deficiency of the sense of sight.

Sense of Hearing.

Auditory aurae of some kind are not uncommon in epilepsy, but not as frequent as visual. In 500 cases there were only eight which may be said to come under the scope

of the present inquiry. These consisted of the sudden and paroxysmal development of a noise, which varied greatly in character, but was generally of a loud hissing or buzzing nature, and was usually present in both ears. Patients find it very difficult to localise these noises, and often describe them as not being in the ears at all, but in the head. These are succeeded by temporary impairment of hearing, also generally on both sides. Sometimes in such cases there is permanent partial deafness. Care, of course, was taken in the instances under notice to exclude any disease of the ear itself. Illustrations of this condition are as follows.

A woman aged twenty has been subject to epileptic attacks for a year. These are always preceded by the sudden development of a loud noise, which is described as being like "the ringing of a bell," and which, it is asserted, is confined to the left ear and side of the head. After this has lasted for an appreciable time, she is seized with a convulsion, always beginning on the left side of the face, and thence extending to the arm and leg, and which lasts some minutes, accompanied all the time by the ringing noise and extreme giddiness. She does not lose consciousness. In a few minutes all this ceases, leaving her weak in the left side and deaf in the left ear. Personal examination with both the watch and fork showed that both ears were deficient in hearing, but the left was undoubtedly the most so. In the course of half an hour the hearing was in the usual condition, substantially normal, perhaps slightly deficient on both sides. These attacks occur several times a week. I had an opportunity of being present at three of these, on all of which the foregoing auditory phenomena were observed. The patient is otherwise well and intelligent, and there is no optic neuritis. This case is unusual, inasmuch as the subjective

noise and subsequent deafness were almost, though not entirely, confined to one side.

A woman aged forty-five has for many years suffered from epilepsy, the fits being accompanied with loss of consciousness and general convulsions. Each is preceded by a loud "buzzing" noise in the head. After the attack for some hours the patient is almost quite deaf with both ears, so that she "cannot hear what is said to her."

A man aged forty-eight, the same who had the visual aura followed by homonymous hemianopia, is permanently quite deaf with the right ear. Preceding his attacks, in addition to the flashes of light, he experiences a loud "buzzing" in his ears, especially the right, and afterwards he is deafer than usual with the left ear for an hour or so.

In some cases, when noises in the ear are complained of as preceding the attacks, there is said to be no deafness afterwards. Possibly this is not sufficient to be markedly noticed. In no other cases except those described have I personally had an opportunity of actually testing this phenomenon, for in this, as in all cases of epilepsy, it is only by chance that the attacks take place under observation. So far as they go, these observations indicate that the sense of hearing follows the same laws as those of touch and sight.

Sense of Taste.

An aura limited to the sense of taste is rare. Of 500 cases of epilepsy, in only four was this a symptom. For information on three of these I am dependent on the statements of the patients. The only one which I had an opportunity of personally investigating is sufficiently definite, and constitutes a valuable observation.

A woman aged thirty has for some years been liable to convulsive seizures, generally of the left side. These are sometimes accompanied by loss of consciousness, sometimes

not. The first symptom of an attack is the sudden experience of a nauseous taste in the mouth. This is described as "putrid," at other times as "coppery." Then she usually loses consciousness and has a convulsion. On recovering from this she says that she is "unable to taste anything" for the rest of the day, and cannot tell one kind of food from another. She also asserts that her powers of smell are deficient. Occasionally the gustatory aura takes place alone without loss of consciousness. I was present when this woman had one of her attacks. Suddenly she became pale, looked vacant, and sat down. She said "I feel the taste." She made a very wry face, and said on inquiry that it was "like rotten eggs." She smacked her lips and swallowed saliva, which seemed to flow in larger quantities than natural. She gulped at the throat, which action was accompanied by a noise. This lasted for a minute or so, the patient feeling faint and giddy, but she did not lose consciousness, and there were no convulsions. Then the sensation of taste passed off and she recovered her usual condition, with the exception of a headache which remained. On testing with a variety of substances, there was distinct evidence that the sense of taste was almost entirely absent. She could not tell salt from sugar. Half a teaspoonful of salt was put in the mouth and she did not know what it was. Only when masticated and swallowed she said she "thought it was salt." The same occurred with sugar. Ordinary food, such as bread, she did not taste at all. It was impossible to determine whether this deficiency was more marked on one side than the other. My impression is that it was not. This condition continued for the remainder of the day. On the following morning the sense of taste was normal.

A lad aged sixteen, who had had ordinary epilepsy for many years, says that his fits are always preceded by "a

nasty taste in the mouth." He asserts that on recovering consciousness he has lost the sense of taste for the remainder of the day.

A boy aged twelve has a similar aura, but does not think his taste is afterwards affected. This has probably not been noted.

A woman aged twenty-nine says that before all her fits "her mouth waters," and that she has a peculiar "sensation" in her mouth, but has not noticed any after-effects.

This completes my own experience of disturbances of the sense of taste in epilepsy. Such cases are undoubtedly rare, but would be found to be more common if they were looked for. The phenomena observed harmonise with what has been ascertained to take place with the other senses.

Sense of Smell.

Olfactory auræ are also of very rare occurrence. Of 500 cases of epilepsy, I have only found in four that this has been a pronounced symptom. Here also I have only one personal observation, which, however, is of great interest.

A man aged forty has had for two years several seizures every day. First he experiences a disagreeable pungent smell; then there is a spasm of the right side of the mouth which spreads to the arm, and sometimes, but rarely, to the leg of the same side. This lasts a minute or two, without loss of consciousness. It is accompanied by flushing of the face and right ear, which becomes intensely hot. He then recovers. I was present at several of these attacks, and carefully noted the symptoms. Suddenly the patient would say, "I feel the fit coming on." He pursed up his face with a disgusted look, and, when questioned, said, "I have a disgusting smell in my nose." He could not otherwise describe it. He was pale, sat down feeling giddy and faint, but was perfectly intelligent, and there was no loss of senses

or convulsion, and otherwise nothing wrong was to be observed. This smell continued for a minute or two, and then passed off. Shortly after, on testing with all kinds of scents, he was unable to detect any of them in either nostril. This continued for the remainder of the day. Practically this patient was nearly always without the sense of smell, as he had several of these attacks every day; but when under treatment they were diminished in number; it was found that he recovered the power—possibly, however, not to its full extent. This purely olfactory attack by itself was not common with this patient, but was usually accompanied with spasm on the right side. I heard that this man finally died in the infirmary, but I could obtain no further information concerning him.

A boy aged twelve, the same who had a gustatory aura, also had a sensation of smell preceding his attacks. He did not know whether the sense was afterwards lost.

In two other cases a sensation of smell was said to precede ordinary epileptic attacks, but nothing further could be ascertained concerning them, and no opportunity occurred for personally investigating them.

The facts in relation to smell as well as taste are certainly very meagre, as the phenomena are very rare, but the two cases which were carefully observed by myself entirely coincide with the signs which ensued from irritation of other cortical centres.

Summary.—The general conclusions to be derived from the foregoing investigation may be summarised as follows. 1. In a given series of cases of epilepsy or of epileptiform attacks there are a certain percentage in which a pronounced feature consists of an aura of one of the special senses. In 500 cases there were in round numbers an aura of the sense of touch in 10 per cent., of sight in 3 per cent.,

of hearing in 1·5 per cent., of smell in 0·75 per cent., and of taste in 0·75 per cent. 2. These auræ take the form of the development of crude subjective sensations of one of the special senses, and consist of a sudden attack of exaggerated sensation of pain, light, noise, smell, or taste. 3. These exaggerated sensations of sense are immediately succeeded by the opposite condition—namely, by a temporary abolition or diminution of the special sense previously affected, which results in anæsthesia, amblyopia, deafness, loss of taste, and anosmia. 4. These facts seem to indicate that each of the special senses is separately represented in the cerebral cortex, and that each of them is liable to disease. When by irritation they are in a hyper-physiological condition, they discharge their respective functions, the result being a crude subjective sensation of the corresponding sense. When destroyed or exhausted, the function is abolished or temporarily depressed. 5. The sensory cortical centres thus obey the same general laws, in their relations to disease, as do the motor cortical centres.